The approach to the study of epilepsy has been made by various routes—clinical, experimental, pathological, and biochemical. Most recently it has been viewed from the psychological standpoint. Much has been written as to whether epilepsy is based upon an organic foundation or is a functional disorder. It is generally recognized that some cases are symptomatic of an organic lesion of the brain—vascular, traumatic, or neoplastic; others are of toxic origin—alcoholic, eclamptic; others are of infective nature; others are associated with degenerative cardiovascular disease. When all cases of symptomatic epilepsy have been put aside, a large residuum remains belonging to the so-called idiopathic group. This division of the epilepsies, it would appear, may be reduced by the recognition of a psychological series.

In the two works referred to in this review, the attempt has been made, on the one hand (Redlich1) to produce evidence of the essentially organic nature of all epilepsy; and on the other (Clark2) to prove the existence of a definite group of the epilepsies arising from psychogenic causes. It is the object of the reviewer to lay before the reader such salient facts as may be necessary to inform him of the present-day position of the epilepsies as revealed by the study of the works of Redlich and Pierce Clark respectively.

I. Redlich may be regarded as a protagonist of the organic nature of epilepsy. His work is a detailed and elaborate study in which is brought forward all the published evidence that goes to prove the organic character of this disease. It might perhaps be desirable to state his general conclusion before referring in some detail to the evidence on which it is based. He says it is difficult to classify epilepsy as a pure neurosis in view of the picture presented of its chronic course, its severe fits, and its symptoms both in the somatic and psychical spheres. He places chronic epilepsy amongst anatomico-pathological disorders, and calls attention to the diffuse changes which are found in the brain both in the so-called genuine disease and in the
obviously organic forms as evidence of the 'oneness' of these disorders. In his view the term 'genuine epilepsy' should be allowed to lapse, because from all sides it is impossible to define with certainty a group to which this term may legitimately be applied.

Much divergence of opinion has existed as to what should be regarded as genuine epilepsy. Reynolds held as genuine only those cases which occurred without structural changes in the brain and without disease of other organs; Binswanger only those chronic cases with a predisposition or congenital epileptic constitution; Gruhlé only those in which the etiology was uncertain; Gélineau only those in whom no cause could be found. It is generally agreed, however, that a chronic course and a tendency towards permanent psychical changes are essential for the diagnosis of genuine epilepsy.

We are confronted with the same difficulty if the time of onset is taken as a basis, for it is often difficult to determine the time at which the epilepsy commences. A large number of cases of chronic epilepsy begin in early life. A few fits may occur in childhood and then remit, to return at puberty or even as a late epilepsy. Other cases begin in an infantile eclampsia, sometimes associated with infective causes, but do not always go on to a chronic epilepsy. Some infantile convulsions are due to encephalitis, which may give rise to epilepsy later in life.

A like difficulty arises over the late epilepsies. Quite a number of these cases have a direct heredity to epilepsy; others may have had convulsions in infancy or a few fits about puberty, which cease until arteriosclerotic changes favour their recurrence in permanent form. The late epilepsies are more often associated with organic lesions of the brain and nervous system than the early epilepsies, but etiologically it is not right to separate the late from the so-called genuine variety, as there are late epilepsies without anatomical lesions just as there are early epilepsies with them.

If we turn to the exciting causes of epilepsy we find a large number of cases presenting infective, toxic, traumatic, neoplastic, and other agencies at work in the production of the disease. The influence of acute infective disease, especially scarlet fever, in the causation of epilepsy is well known. This disease may originate an epilepsy through toxic agencies or by the production of organic lesions of an encephalomeningeal character. Syphilitic infections also may lead to a neurosyphilitic variety of epilepsy, sometimes of toxic origin, at other times of meningeal or vascular origin. Redlich holds that all forms of neurosyphilis offer great possibilities for epilepsy in view of the many organic lesions which occur in the brain in consequence of this infection. The effect of pregnancy and the puerperium upon epilepsy is important. Epilepsy may be latent during a pregnancy.
The development of epilepsy out of a puerperal eclampsia has long been recognized. It has been thought that in this type of the disease a toxico-infective cause is at work. On the other hand a 'pregnancy epilepsy' may temporarily subside, but recur and eventually become a chronic epilepsy of the menstrual type. It is suggested that in the 'menstrual type' of epilepsy, which is common at the outset of many cases of chronic epilepsy, a chemical influence is at work through the agency of ovarian hormones.

The relations between epilepsy and endocrine function are uncertain. Redlich maintains that the organs of internal secretion play a not unimportant part in the production of epileptic seizures. The association between hyperthyroidism and epilepsy in young women is not unusual, but whether as cause and effect is not clear. The relation between epilepsy and tumour of the pituitary body also is not uncommon. Here the epilepsy may be the first symptom, or the symptoms of pituitary tumour may ensue during the course of a chronic epilepsy. Reference also is made to the action of the parathyroids in tetany and epilepsy. The whole subject, however, of the influence of endocrine secretion—normal or abnormal—requires fuller investigation in its relation to epilepsy.

If we turn now to symptomatology, it is argued that there is no essential difference between the fits of organic epilepsy and those of the so-called genuine disease in their clinical expressions. An organic lesion of the cerebral cortex gives rise usually to fits of the focal or Jacksonian type; but, if the lesion is subcortical in position, although one side of the body may be affected in excess of the other, the fit itself may have the common features of an epileptic seizure. It is recognized as an accepted principle that the seizures occurring, for example, in a case of cerebral tumour involving the frontal or the temporosphenoidal lobes may have all those features common to the reactions of genuine epilepsy, such as various forms of minor epileptic attacks, twitchings and spasms of head and eyes, limbs, or body, psychical attacks and general convulsions, with or without warning.

Typical epileptic fits with post-convulsive psychoses may run a chronic course for many years before localizing or general symptoms of intracranial tumour make their appearance. There may be, indeed, single features by which the fits of genuine epilepsy may be differentiated from those of organic epilepsy, but they are of a secondary nature. The post-convulsive paralyses are not constant, nor are they entirely characteristic, and the Babinski plantar response may be obtained temporarily after a severe fit, or may persist for a time after status epilepticus or the status hemi-epilepticus. Redlich admits that post-convulsive signs are not common in genuine epilepsy and are often only of the slightest character. They are more common
after frequent and severe fits, and indicate the summation of ‘exhaustion’ symptoms. In his view these sequelae are associated with histological changes in the brain, and their persistence indicates an increase in these changes, probably of the nature of a gliosis.

He argues also that transitional forms may be found in a study of epilepsy with infantile cerebral paralysis. Thus a latent infantile cerebral palsy may be made manifest by a fit. The only means by which these seizures may be distinguished from those of genuine epilepsy is by their more unilateral character. The lesion may be focal or diffuse, cortical or subcortical. The original palsy may have disappeared, the history alone denoting its previous existence, although, if the lesion was in the left hemisphere, left-handedness may be present.

In like fashion the existence of psychical symptoms in epilepsy and of psychical epileptic equivalents offers no argument against the organic nature of this disease, as they are observed in cases of epilepsy associated with tumor cerebri, meningitis, and multiple sclerosis. A large number of cases of chronic epilepsy show some degree of mental enfeeblement or dementia, which is to some extent dependent upon the frequency, severity, and quality of the fits. According to Redlich, this characteristic epileptic dementia is based upon extensive anatomico-pathological changes in the brain. The organic epilepsies of early childhood may be associated with a profound degree of feeble-mindedness often bordering on imbecility, which may be further augmented by the attacks. On these grounds it would seem to be impossible to draw a distinction between the so-called genuine epilepsy and that associated with infantile cerebral paralysis.

Pathological lesions of all kinds abound in cases of epilepsy with or without infantile cerebral palsy. Such are cysts, cystic collections of fluid in the meninges, thickening of the meninges, varicosity of certain veins, diffuse sclerotic and meningeal changes following acute infective diseases and hydrocephalus.

In view of such organic findings, may not the so-called genuine epilepsy have a pathological basis? Sclerosis of the cornu Ammonis is found in from 30 to 60 per cent of all cases of epilepsy, and a ‘Rand-gliose’ is not infrequent. Morbid changes in the brain of an acute character have been observed and studied in consequence of severe fits and the status epilepticus. Redlich regards these changes less as due to the fits than as the anatomical correlation of the process which evokes the fits.

Taking all these facts into consideration, Redlich concludes that it is difficult to regard epilepsy as a pure neurosis. He believes that the pathological changes which are found in the organic epilepsies and in the so-called genuine disease are evidence of the ‘oneness’ of these disorders.
II. In *Clinical Studies* Pierce Clark has elaborated in great detail the psychogenic attitude towards a group of epilepsies. I recommend the study of Clark's work on this subject to those who are interested especially in the psychological investigation of epilepsy. One feature stands out with notable prominence, viz., the value of a meticulous examination of the psychological history and temperamental make-up of the epileptic, both actual and potential.

As the *Psychiatric Bulletin* is not obtainable in our medical libraries, I shall give a précis of this work in order to bring before the reader the main facts bearing upon the psychogenic origin of epilepsy.

The psychological study of idiopathic epilepsy includes the recognition of the temperamental make-up of the epileptic, and the analysis of the mental content accompanying the convulsive or other characteristic reaction. It is held that the temperament in epileptics is important, as it is this quality of mind which determines the adaptational failure and the development of the seizures or other equivalent reactions of epilepsy.

This temperament may be stated briefly to be characterized by egotism, morbid sensitiveness, and poverty of ideas. Epileptics invariably are self-opinionated, conceited, and self-assured. They are often moody, with periods of lethargy, alternating with outbursts of hastiness. They are difficult to live with. They adapt themselves badly to social conditions, become self-centred, morbid, and asocial.

In consequence of these defects the epileptic is incapable of social adaptation and is rendered inadequate to lead a normal adult life. This temperamental condition may be studied even in childhood, and may be well marked many years antecedent to the onset of the fits. Long before the attacks of epilepsy develop, other signs of mal-adaptation may be observed in day-dreams, outbursts of temper, irritability, and depressive phases.

Thus it comes about that when the new and elaborate adaptations required at puberty and adolescence have to be met, the temperamental deficiencies are such that failure occurs, and characteristic reactions are the direct outcome of the inability of the subject to subordinate individualistic tendencies to social demands.

Pierce Clark maintains that the epileptic seizure is a reaction away from stressful reality, and may serve some useful purpose by enabling the patient to escape from responsibility or avoid some necessity for adaptation.

The reactions are not always similar. There are the common forms of petit mal as well as the major epileptic seizure, and there are the various epileptic equivalent reactions, such as abstractions, lethargies, outbursts of uncontrollable excitement and temper, depression, mania, and the like.
A mental exploration or psychological analysis of epileptics may bring to the surface all kinds of mental conflicts and repressions, which favour the onset of the epileptic reactions in consequence of the inability on the part of the patient to adjust his outlook to particular circumstances, such as periods of stress or disappointment.

The nature of the stress may vary. It may be an inability to adapt to school or college work or to the disappointments of a business career. It may be fatigue or physical strain. It may be of the nature of a mental conflict, with associated repressions. If the subject can be kept free of irritation, under moderate adjustments of work, he may continue free of attacks. Indeed, the actual convulsive seizure may be only the most severe reaction in a whole series of maladjustments. Thus a careful daily study of the epileptic may show great variability in the mental state; a period of irritability may be followed by one of spontaneous interest; a phase of cheerfulness may be succeeded by one in which 'nothing is right'; upon these a major epileptic fit may supervene, to be followed by a spell of relief and quietness. It is well known that irritability and fault-finding lasting for a day or two may be succeeded by a convulsive seizure at night which 'clears the air' for a while.

It is held that the real motivation of the fit may be unconscious, while that which would appear to be sufficient may not be followed by an attack. It would appear also that the ordinary immediate stimulus of the fit is not the irritation itself, but the effort of repression not to respond to the irritative stress which the patient feels and which, if allowed expression, might be violent and excessive.

It is well known that parents who suppress the 'tantrums' of an epileptic child find that such suppression may provoke more frequent and severe fits. Repression may increase nervous tension to the point that a convulsive reaction may be the means of temporarily withdrawing the person from a too demanding environment. It may be assumed that the disorder in genuine epilepsy has to do with adjustments at deep instinctive levels of the nervous system, and that the fit as a reaction to failure may be so severe as to threaten even life itself.

It is obviously impossible to conduct a psychological analysis during the period of unconsciousness, but an examination of this character may be made during the milder types of seizure, in the transitory confusion accompanying minor attacks, and in the post-convulsive dream states of the major attack. A study also of the epileptic deliria and the spontaneous remarks which are made by patients in these conditions, if noted and analyzed, may throw light upon the mental conflict underlying a particular epileptic reaction.

Usually three sets of psychic events have to be noted. First, the remote and immediate stresses which aggravate and promote the
occurrence of individual epileptic reactions; secondly, the actual mental content of the specific attack; and thirdly, the early and ultimate free association upon the essential words or ideas expressed in the content.

Epileptics may be divided into those whose adaptational disability is slight—the high-grade type—and those who are badly adapted, or the chronic type. Amongst the former we find those patients in whom a cure results and in whom efficient treatment may bring about a strengthening of their adaptive power; in the latter, whose temperamental defects are pronounced, a steadily progressive epilepsy develops. The social position of the confirmed epileptic is unsatisfactory, and unfavourable for adaptation. They become the victims of social repression which tends to aggravate the disability. Hence the chronic epileptic loses spontaneity and contact with the world; he becomes apathetic, dull, and anergic. He has, in fact, passed into the condition of permanent renunciation to efficient adjustments to reality, viz., epileptic dementia.

Clark makes some important remarks upon the treatment of epilepsy. He holds that a rational therapy can be based only upon a careful effort to understand the make-up and the mental mechanism in this disorder. Attention must not be restricted to the occurrence of individual seizures. Children predisposed should be taught how to approach the difficulties of a situation. It may be necessary in the young to omit the intellectual side of training and to tutor the patient entirely in social behaviour. The discipline and training at the average school often furnish just that degree of stress which is too much for the epileptic. Hence educative training should be entirely individualistic and elastic. The epileptic requires novelty and wide range of educational appeal, and every effort should be made to adapt the patient along ethical, moral, and social lines. His training and treatment are often carried out best away from home. Special attention and study should be given to his emotional and intellectual interests, and it is necessary to combat the temperamental antisocial tendencies.

Redlich accepts the theory, which has been held for many years by a number of workers upon this subject, that epilepsy is an organic disorder. This view has received support from the unsatisfactory results of treatment, from the large number of epileptics who become victims of chronic epilepsy, and from the progressive character of both the paroxysmal and the interparoxysmal symptoms. It has been confirmed by the acknowledged existence of pathological changes in the brain, although a doubt has been expressed as to whether these changes are the cause or the consequence of the seizures.
According to the psychogenic theory of epilepsy, the seizures and other paroxysmal phenomena are reactions away from stressful reality, and serve a useful purpose by enabling the patient to avoid some necessity for adaptation. The chronic disease denotes well-marked temperamental defects with persistent adaptational failure; while the dementia is due to the crippling effect of frequently recurring types of reaction and denotes a permanent renunciation to effective adjustments.

The psychogenic theory has brought epilepsy into line with other functional nervous disorders attributable to adaptational failure. The tendency is to regard hysterical and epileptic reactions as symptoms of temporary disintegration of functional levels of the nervous system. The hysterical attack is obviously psychogenic in origin, and may exhibit all grades of severity, from a simple hysterical ‘fit’ up to the complex seizure of major hysteria (hystero-epilepsy). In the attack the disturbance of consciousness is superficial and slight. In the epileptic fit, the convulsion passes rapidly over into deeper levels, the muscular reactions are disorganized, and consciousness is early and completely abolished. There would appear to be a ‘border zone’, however, in which it is difficult to say whether the observed attack is of a hystero-epileptic nature or is genuinely epileptic.

It has been stated that emotional shock may lead to attacks in no respect different from those of idiopathic epilepsy; and it is contended that the psychogenic origin of a seizure should not be ignored because in the fit the patient has bitten his tongue or has been incontinent. “We are compelled to recognize that certain patients exhibit seizures clinically indistinguishable from ordinary epilepsy as reactions to situations of a purely psychical nature. In other words, the attacks are due to extraneous psychical causes such as have been supposed to be characteristic of hysterical fits only.”

But the experience of the recent war has thrown a somewhat remarkable light upon this subject. Although the psychoneuroses have shown a largely increased frequency under conditions of mal-adjustment and mental conflict in war experience, genuine epilepsy arising de novo under war conditions has been rare. The majority of epileptic soldiers were epileptic before joining the army, and many so-called epileptic fits in soldiers were in reality hysterical convulsive reactions. It may be agreed that individual seizures may arise from emotional stress or mental conflict in a patient with established epilepsy; but a much fuller understanding of the mechanism of epilepsy and of its root causes is necessary before the purely psychogenic causation of this disease can be accepted, and its treatment by psychotherapy regarded with assurance. In order to establish epilepsy as such, we have to look beyond the seizure or its equivalent
reaction, and to find it in the characteristic temperament or mental make-up, and in the tendency towards mental enfeeblement which invariably follows in the chronic or confirmed disease.

The acceptance of the psychogenic origin of epilepsy obviously affects its treatment as at present conducted. But as the fit itself may form a stumbling-block to satisfactory adaptation, and prove an incubus to a return to health and work, any medicinal remedy which will subdue, minimize, or arrest the seizures may be prescribed. On the other hand, we know that the prevention or postponement of a fit is not always desirable. The fit, in some instances, is so definitely a reaction to a difficult situation, which the patient is unable to meet normally, that its occurrence 'clears the air' and a feeling of relief is the outcome.

The theory of the psychogenic causation of epilepsy therefore has opened up another route along which the study of this intractable disease may again be approached.

REFERENCES.

1 Prof. Dr. E. Redlich, Die klinische Stellung der sogenannten genuinen Epilepsie. Berlin, 1913.


3 Annotation, Lancet, 1919, ii. 791.